WHAT IS CLAIMED IS:

1. A method for screening for drugs for the treatment of Alzheimer's disease, said method comprising:

contacting mutant hippocampal cells having enhanced synaptic potentiation upon stimulation as compared to wild-type hippocampal cells with a candidate drug;

subjecting said mutant cells to tetanic stimulation; and

determining the effect of said agent on the synaptic potentiation of said mutant hippocampal cells;

wherein a reduction in the enhanced synaptic potentiation of the mutant hippocampal cells is indicative of activity of a candidate drug for the treatment of Alzheimer's disease.

2. A method according to Claim 1, wherein said mutant cells are mutated in a presenilin gene.

3. A method according to Claim 2, wherein said mutant cells are mouse hippocampal tissue slices.

4. A method according to Claim 1, wherein said enhanced synaptic potentiation is as a result of a change in the $GABA_A$ receptor pathway.

5. A method for screening for drugs for the treatment of Alzheimer's disease, said method comprising:

contacting mutant hippocampal cells, having enhanced synaptic potentiation upon stimulation as compared to wild-type hippocampal cells, with a candidate drug;

subjecting said mutant and wild-type hippocampal cells to a tetanic stimulus;

measuring changes in potentiation with time of the mutant and wild-type hippocampal cells and comparing the effect of said agent on the synaptic potentiation of said mutant as compared to the observed synaptic potentiation of said wild-type hippocampal cells;

wherein a reduction in the enhanced synaptic potentiation of the mutant hippocampal cells as compared to the synaptic potentiation of the wild-type cells is indicative of activity of a candidate drug for the treatment of Alzheimer's disease.

6. A method according to Claim 5, including the additional steps of:

contacting mutant hippocampal cells having enhanced synaptic potentiation upon tetanic stimulation as compared to wild-type hippocampal cells with a $GABA_A$ receptor antagonist;

subjecting said mutant and wild-type hippocampal cells to tetanic stimulation; and

measuring changes in synaptic potentiation with time of the mutant and wild-type hippocampal cells and comparing the effect of said $GABA_A$ receptor antagonist on said mutant and said wild-type hippocampal cells;

wherein a reduction in the enhanced synaptic potentiation of the mutant hippocampal cells without a significant change in the synaptic potentiation of the wild-type cells is indicative of the mutation acting on a common pathway with said $GABA_A$ receptor antagonist.

7. A method according to Claim 5, wherein said agent is present with said wild-type hippocampal cells.

8. A method for screening for drugs for the treatment of Alzheimer's disease, said method comprising:

contacting mutant hippocampal cells having enhanced synaptic potentiation upon stimulation as compared to wild-type hippocampal cells with a candidate drug;

subjecting said mutant and wild-type hippocampal cells to a tetanic stimulus at a first potential of glutamate currents and a second potential of $GABA_A$ currents;

measuring the synaptic response at each of the first and second potentials for the mutant and wild-type hippocampal cells and comparing the effect of said agent on said mutant and said wild-type hippocampal cells;

wherein a reduction in the enhanced synaptic response of the mutant hippocampal cells without a significant change in the synaptic response of the wild-type cells is indicative of activity of a candidate drug for the treatment of Alzheimer's disease.

9. A method for screening for drugs for the treatment of Alzheimer's disease, said method comprising:

contacting mutant mouse hippocampal cells mutated in the presenilin-1 gene and having enhanced synaptic potentiation upon tetanic stimulation as compared to wild-type hippocampal cells with a candidate drug;

subjecting said mutant and wild-type hippocampal cells to tetanic stimulation; and

comparing the effect of said agent on said mutant and said wild-type hippocampal cells upon tetanic stimulation;

wherein a reduction in the enhanced synaptic potentiation of the mutant hippocampal cells without a significant change in the synaptic potentiation of the wild-type cells is indicative of activity of a candidate drug for the treatment of Alzheimer's disease.

10. Slices of mouse hippocampal cells having a mutation in a presenilin gene combined with a candidate drug.

11. Slices of mouse hippocampal cells according to Claim 10, after tetanic stimulation.

12. Slices of mouse hippocampal cells according to Claim 10, wherein said mutation is the PS-1 Δ9 mutation.

add a3